

CHOLINERGIC MUSCARINIC RECEPTOR SIGNALING MODULATES COLONIC INTESTINAL STEM CELL ACTIVITY AND DIFFERENTIATION

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Poster

Colorectal

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Genetic ablation studies in mice reveal regional differences in how muscarinic cholinergic receptors M3R and M1R regulate intestinal stem cells, with M1R playing a more prominent role in colonic stem cell modulation than M3R.

KEY POINTS

- M3R ablation in mouse small intestine reduced Lgr5+ intestinal stem cells and triggered compensatory expansion of DCLK1+ tuft cells, but this pattern did not occur in the colon of the same mice
- In colonic tissue, M3R ablation reduced both Lgr5+ stem cells and DCLK1+ tuft cells while expanding p-EGFR+ tuft cells, suggesting regional differences in how stem cell subtypes sense cholinergic signaling interruption
- Colonic epithelium expressed Chrm1 (M1R gene) more prominently than Chrm3 (M3R gene), and M1R ablation produced more pronounced changes to differentiation and Lgr5+ stem cells than M3R ablation in colon
- M3R ablation upregulated PI3K signaling, which was abrogated by combined M3R and M1R ablation, indicating the two receptor subtypes modulate distinct intracellular pathways
- This work is relevant to researchers studying enteric nervous system regulation of intestinal epithelial homeostasis and regeneration in different intestinal regions

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